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Left ventricular-arterial coupling in septic shock: A physiological review

Juan Daniel Caicedo Ruiz [a,b], Jose Luis Aldana [a,b], Eduardo Kattan [c], Nicolás Orozco [a,b], Gustavo García Gallardo [a,b], Jorge Iván Alvarado Sánchez [d], Juan José Diaztagle Fernández [e,f], Jihad Mallat [g,h,i], Gustavo Adolfo Ospina-Tascón [a,b,*]

[a] Department of Intensive Care Medicine, Fundación Valle del Lili, Cali, Colombia
[b] Translational Research Laboratory in Critical Care Medicine (TransLab-CCM), Universidad Icesi, Cali, Colombia
[c] Departamento de Medicina Intensiva, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile
[d] Department of Intensive Care, Fundación Santa Fe de Bogotá, Bogotá, Colombia
[e] Department of Physiological Sciences, Faculty of Medicine, Universidad Nacional de Colombia, Bogotá, Colombia
[f] Fundación Universitaria de Ciencias de la Salud, Bogotá, Colombia. Department of Internal Medicine, Hospital de San José, Bogotá, Colombia
[g] Critical Care Division, Integrated Hospital Care Institute, Cleveland Clinic Abu Dhabi, Abu Dhabi, United Arab Emirates
[h] Cleveland Clinic Lerner College of Medicine of Case Western Reserve University, Cleveland, OH, USA
[i] Faculty of Medicine, University of Banja Luka, Banja Luka, Republic of Srpska, Bosnia and Herzegovina

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ABSTRACT

Conventional modeling of cardiovascular function prioritizes the interplay between cardiac contractility and venous return curves as primary determinants of stroke volume. However, this approach does not capture the influence of arterial load in flow generation, thus hindering a more comprehensive understanding of macrovascular dynamics. Since both heart and arteries are elastic chambers with distinctive geometries housing blood volume, their coupling could be derived from pressure-volume curves, which ultimately denote time-varying elastance relationships. Accordingly, the left ventricular-arterial coupling (LVAC), represented by the ratio between arterial and ventricular elastances, could indirectly estimate the energy transmission from the heart to the proximal arterial tree, thus reflecting cardiovascular efficiency. Assessment of LVAC by combining bedside echocardiography and arterial pressure monitoring may improve the understanding of macrohemodynamics during shock conditions and might potentially help to evaluate the impact of cardiovascular therapies. Nevertheless, cardiovascular dysfunction in septic shock result from complex interactions and highly variable degrees of hypovolemia, pathological vasodilation, peripheral vascular decoupling, microvascular blood flow misdistribution, and heart-pump dysfunction, whereby optimizing LVAC during the resuscitation process would not necessarily reverse tissue hypoperfusion. This review provides an analysis of the components of LVAC, including the physiological fundamentals of arterial and ventricular elastances, explores its relationship with cardiovascular efficiency and energetics, synthesizes available data about LVAC assessment in septic shock, addresses some unresolved controversies, and proposes a conceptual framework for future research.

1. Background

Classic models describing cardiovascular function emphasize on the interplay between cardiac contractility and venous return curves as the main determinants of the stroke volume [1,2]. Nevertheless, this vision underestimates the role of energy transmission from the heart to the vascular periphery and the effects of arterial load on the blood flow generation. The heart might be conceptualized as a flow generator that operates in conjunction with the arterial afterload, i.e., the mechanical opposition that the heart faces when pumping blood, namely the arterial impedance [3]. Although the total flow generated by the cardiovascular system is ultimately driven by tissue metabolic demands [4], ventricular mechanical efficiency is basically determined by the interaction between the cardiac contractility and the arterial impedance [5–7]. This dynamic interplay between myocardial contractility and vascular load defines the concept of left ventricular–arterial coupling (LVAC).

* Corresponding author at: Department of Intensive Care Medicine, Fundación Valle del Lili, Translational Research Laboratory in Critical Care Medicine (TransLab – CCM), Universidad Icesi, Cra. 98 #18 – 49, 760031 Cali, Colombia.
E-mail addresses: gaospina@icesi.edu.co, gusospin@gmail.com (G.A. Ospina-Tascón).

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LVAC depicts the relationship between the arterial load conditions estimated by the arterial elastance (Ea) and the heart contractility estimated by the end-systolic elastance (Ees). The Ea/Ees ratio, a quantitative representation of LVAC, provides a dimensionless value that can be illustrated on the pressure–volume diagram [9] (Fig. 1), and that ultimately reflects the energy transmitted from the heart to the proximal vascular tree. In healthy individuals, values of LVAC (i.e., Ea/Ees ratio) should be slightly less than 1.0, indicating that, under physiological conditions, the cardiovascular system optimizes energy expenditure [5,6,10]. In contrast, LVAC values often exceed 1.0 during shock states [11–13], reflecting impaired cardiac contractility and/or relatively increased load conditions, a situation denoted as left ventricular-arterial uncoupling. This review aims to provide a physiological understanding of LVAC, detailing its components (Ea and Ees), describing methods used for its estimation, and outlining the physiological bases that potentially supports non-invasive LVAC assessment to monitor cardiovascular hemodynamics.

2. Arterial elastance: the physiological concept

2.1. The origin of the arterial load

Load might be defined as the restriction to the flow generated by a hydraulic pump, that consequently pressurizes the system [14,15]. The physiological concept of arterial load encompasses both resistive and pulsatile components. The resistive component comprises the hydraulic forces opposing to the non-pulsatile flow and represents the bulk of arterial load (80–90%) [16]. Meanwhile, the pulsatile component arises from the intermittent contractile activity of the heart and the inability of the arterial system to fully dampen the oscillations generated by the cyclic ventricular ejection. Remarkably, under physiological conditions, contribution of the pulsatile component to the hydraulic load is low (10–20%) because of the high distensibility of the proximal aorta [1,16]. The sum of the resistive and pulsatile components of arterial load represents the impedance (Z) of the vascular system. Therefore, the arterial load faced by the heart might be quantified from the estimation of Z at the aortic level (namely, the input impedance). Z is a complex number comprised of magnitude (real) and phase (imaginary) components, which could be obtained by frequency analysis of simultaneous pressure ($\overline{P}$) and flow ($\overline{Q}$) over a single, or multiple heartbeats. Hence, the oscillating phenomena of $\overline{P}$ and $\overline{Q}$, represented in Eq. 1, denotes the magnitude (or absolute value) of Z [1,13].

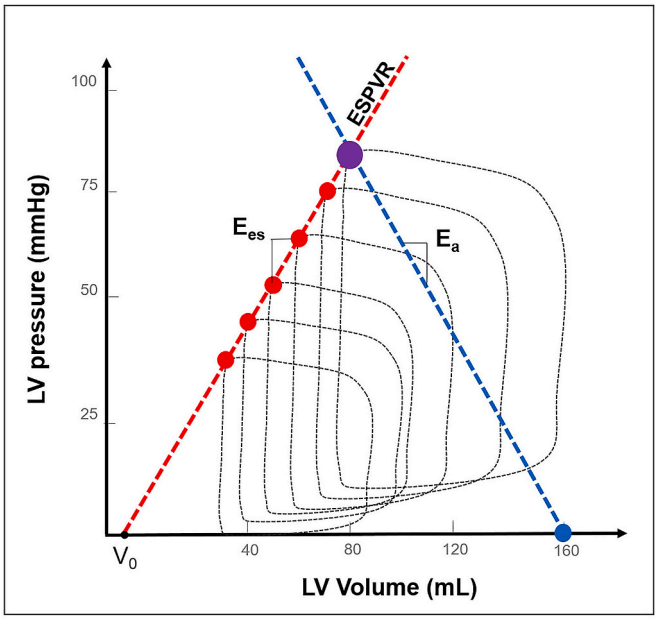

Fig. 1. Left ventricular pressure – volume (P—V) diagram and the left ventricular-arterial coupling (LVAC). Ees (red line) represents the left ventricular end-systolic elastance, corresponding to the slope of the end-systolic pressure-volume relationship (ESPVR). ESPVR is obtained from multiple P—V loops acquired at different load conditions. V_0 represents the ventricular dead space volume, which corresponds to the theoretical left ventricular volume at zero pressure. Ea (blue-dotted line) represents arterial elastance, which is depicted by the slope of the line originated at the coordinated point composed by end-diastolic volume and zero pressure (blue point) and connects with the coordinated point composed by the end-systolic volume and end-systolic pressure. The point at which Ees intersects Ea (purple point) depicts the LVAC and represents the mechanical efficiency at which the stroke volume is ejected. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

$$|Z| = \frac{\overline{P}}{\overline{Q}} \quad (1)$$

The vascular impedance then provides insights about the arterial structure and function. It is, intrinsically, a magnitude in the frequency domain that characterizes the opposition to pulsatile flow by quantifying the level of storage and dissipation of the hydraulic energy.

2.2. Estimation of arterial impedance by using the Fourier transform analysis

Acording to the Fourier theorem, a periodic function may be expressed as the sum of a series of sine or cosine terms. Thus, any periodic signal can be decomposed into a series of sine waves (also called harmonics) with increasing frequencies as multiples of the original ground frequency signal (in this case, the heart rate). Since arterial pressure and flow waves are of an oscillatory nature under cardiovascular steady state conditions, quantification of impedance (Z) becomes possible by transforming such pressure and flow waves into multiple superimposed sine waves or harmonics, using the Fourier transform. The ratio of pressure to flow of the corresponding harmonic yields the impedance (Z) for those frequencies present in the spectrum, with lower and higher harmonics corresponding to lower and higher frequencies of Z, respectively [1,15,17].

The Fourier analysis allows the decomposition of Z into two key components in the frequency domain: the magnitude (|Z|) and the phase (θ) (Fig. 2, Panel A). The magnitude |Z|represents the absolute value of impedance at each frequency, quantifying the total opposition to pulsatile flow regardless of the delay between pressure and flow waves. Meanwhile, the phase (θ) reflects the direction of the delay between such waves, where $\theta < 0$ implies that the phase lag of pressure is larger than the phase lag of flow, so flow is ahead of pressure. Such behavior indicates a system that behaves as a capacitor, where flow is needed to load the capacitance and build up pressure. Meanwhile, $\theta > 0$ implies that the phase lag of pressure is less than the phase lag of flow, so pressure is ahead of flow. Such behavior indicates a system where inertia dominates, and where a certain pressure build-up is required to generate the flow. Together, all these components describe whether the cardiovascular system predominantly behaves like a capacitor or an inductor at a given frequency [1,15] (Fig. 2, Panel A).

The Fourier transform then provides a detailed analysis of Z. Nevertheless, assessment of Z by using this method in clinical practice is not straightforward since it implies simultaneous quantification of pressures and flows in a determined arterial vessel. Furthermore, evaluation of Z in different frequency domains complicates its interpretation at the bedside. As a result, approaches estimating arterial load often prioritize simplified physiological models in which Ea (as surrogate of Z) is assessed in the time domain.

2.3. The Windkessel model and the calculation of impedance

The Windkessel model, introduced by Otto Frank in 1899, derives its

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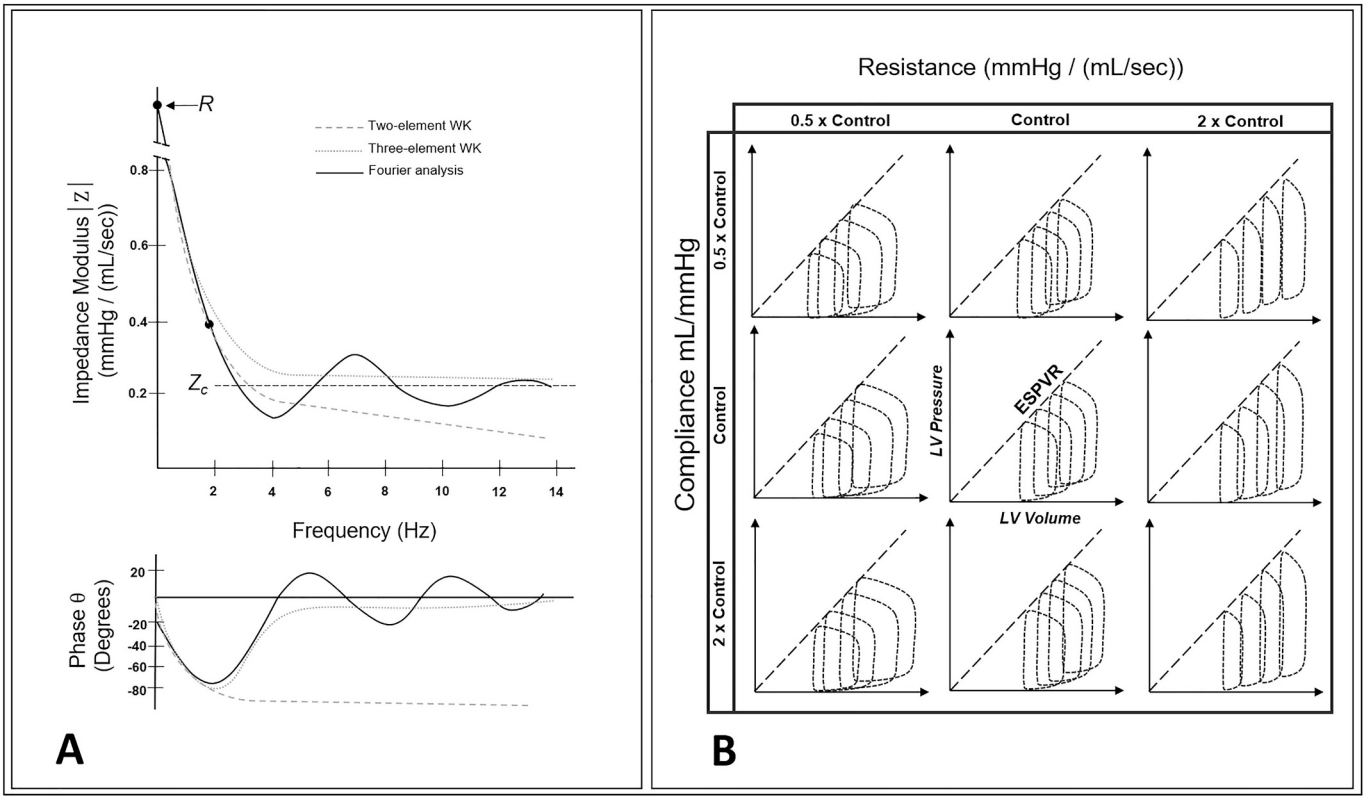

Fig. 2. Impedance spectra obtained by Fourier analysis (two lumped Windkessel model) and P—V loops at variable compliances and resistances.
Panel A. The impedance modulus (|Z|) and phase (θ) are plotted as a function of frequency. The value of |Z| at a frequency of zero corresponds to the systemic vascular resistance (R). As frequency increases, |Z| gradually decreases, reaching its minimum where the phase (θ) crosses zero. This intermediate frequency region of |Z| is strongly influenced by arterial compliance, reflecting the ability of arteries to store and release blood during pulsatile flow. At higher frequencies, |Z| approaches the characteristic impedance (Zc), which is determined by the physical properties of the arterial system, including arterial wall stiffness and blood density. Adapted from Westerhof et al. [19].
Panel B. Multiple P—V loops plotted at different loading conditions. Each plot shows 4 different end-diastolic volumes with different combinations of compliances and resistances. Adapted from Sunagawa et al. [8].

name from the air column used to propel water continuously from a cyclic hydraulic pump [18]. In its early conceptualization, the model comprised only two primary components: (i) a proximal component, which dictates the compliance of the system (C), which is primarily represented by the elastic properties of the aorta; and, (ii) a distal component, represented by arterioles and capillaries, and that predominantly determine the resistance of the system (R). Theoretically, the interplay between these elements would elucidate how the cardiovascular system sustains a relatively constant flow during the diastole. However, the oversimplification of cardiovascular function into these only two components (R and C) proved to be insufficient to fully capture the complexity underlying the total impedance of the vascular system [19]. This limitation prompted the modification of the original model by introducing a third element: the characteristic impedance (Z_C), defined as the ratio of harmonics of the incident pressure to the incident flow. Z_C can be quantified by the input impedance in the absence of wave reflections, and it is usually estimated by the arithmetic mean of |Z| values at frequencies greater than 2 Hz. Importantly, since Z_C excludes the information of wave reflections, Z_C is independent of the heart rate and the properties of the downstream vascular beds. This additional component accounts for the acceleration of blood flow at the onset of systole and is defined by the physical properties of the aortic root. Incorporation of Z_C into the three-element Windkessel model allowed a better approximation of Z. Unfortunately, due to the branching nature and spatially varying elastic properties of the arterial tree, a straightforward computation of Z_C from measured hemodynamic data is not possible. Instead of that, Z_C has to be inferred either from the input impedance spectrum at high frequencies (where reflections have less impact), or from pressure and flow data measured during early ejection phase before the reflected waves have returned to the point of measurement. Nevertheless, limitations to measure Z_C has been overcome by more simple approaches which will be discussed below.

2.4. Arterial elastance as an approximation of impedance (Z)

Following the three-element Windkessel model, Sunagawa and colleagues proposed the effective arterial elastance (Ea) as a surrogate of Z in the time domain [8]. Since the elastance concept encompasses the relationships between pressures and volumes, Ea might be considered as a representation of external conditions opposing to the ejection of stroke volume (SV) [8]. Indeed, Ea might be depicted through the relationship between left ventricular end-systolic pressure (Pes) and SV (Eq. 2) (see Appendix A). Thus, Ea serves as an integrative parameter that encompasses the main components of arterial load: (i) the resistive element; (ii) the total arterial compliance; and (iii) the characteristic impedance. Consequently, quantifying Z in terms of Ea allows the expression of Z through pressure-volume (P—V) relationships in a simpler way in the time domain, without using the Fourier analysis or frequency domain representations [20]. Thus, Ea might be easily expressed as:

$$Ea = \frac{Pes}{SV} \quad (2)$$

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3. End-systolic elastance

3.1. End-systolic elastance and the P—V loops

The concept of Ees introduced by Suga and colleagues [21] relies on the independence of ventricular contractility from load conditions (i.e., both pre and afterload) [21–24]. Early experimental observations suggested that the slope of the end-systolic P—V relationship (ESPVR) is influenced by variations in the intrinsic ventricular contractility. Remarkably, the end-systolic pressure-volume point of any cardiac cycle lies along the same ESPVR over a wide range of load states [2], as demonstrated by Sunagawa and coworkers [8], who corroborated the independence between load and ESPVR when the left ventricular outflow tract from an ex-vivo canine heart was connected to a servo-controlled machine to recreate variable load conditions while recording P—V curves by using pressure – conductance catheters [8].

The slope of the left ESPVR remains essentially constant through multiple combinations of end-diastolic volumes (EDVs), resistances, and compliances (Fig. 2, Panel B). Thus, the slope of the left ESPVR closely reflects left ventricular contractility and acquires the label of end-systolic elastance (Ees) as it represents the relationship between concurrent variations in left ventricular end-systolic pressures and volumes. Ees might be then estimated as the ratio between Pes and the difference between the end-systolic volume (ESV) and the volume remaining at the axis intercept of the ESPVR (which denotes the ventricular volume at zero pressure, i.e., the Vo) (Eq. 3, Fig. 1).

$$Ees = Pes/[ESV - V_0] \quad (3)$$

Unfortunately, direct estimation of Ees implies an intricate and cumbersome procedure in which multiple ventricular P—V loops are acquired at different end-diastolic volumes (EDVs), typically by reducing venous return through balloon occlusion of the inferior vena cava (Fig. 2, Panel B). Furthermore, estimation of Ees entails a precise quantification of left intraventricular pressures and volumes through invasive impedance catheters, which makes this method impractical for being routinely used in clinical practice. This is why non-invasive methods to estimate Ees by using left ventricular single-beat analysis without manipulating load conditions have gained greater popularity.

3.2. Estimation of end-systolic elastance by non-invasive single-beat methods

Multiple techniques have been used to estimate Ees without modifying load conditions. These estimate LVAC during one cardiac cycle and are collectively called Ees single beat ($Ees_{(SB)}$) methods, although some of them still require simultaneous intraventricular pressure and volume quantification [25,26]. Non-invasive methods estimating $Ees_{(SB)}$ are based on mathematical approximations of the behavior of left ventricular elastance in the time domain ($E_{(t)}$), which allow estimating Ees by adjusting variables that render $E_{(t)}$ independent of load conditions [27,28] (see Appendix B). Among them, echocardiographic assessment of $Ees_{(SB)}$ is perhaps the most frequently used in the clinical setting. This approach, introduced by Chen and colleagues [28], evaluates the behavior of $E_{(t)}$ as a function of relatively simple parameters: pre- and ejection times, systolic and diastolic aortic pressures, stroke volume, and left ventricular end-systolic and diastolic volumes (see Supplementary Material Appendix C: echocardiographic assessment of LVAC). Remarkably, this approach assumes that $E_{(t)}$ remains constant during isovolumetric contraction regardless of the load conditions [28]. Unfortunately, Ees assessed by this method might be somewhat inaccurate when compared to other $Ees_{(SB)}$ estimates [29,30]. Nevertheless, despite its limitations, a recent consensus document on the role of ventricular–arterial coupling assessment in cardiac disease and heart failure accepts, and indeed, recommends the non-invasive echocardiographic approach for estimating Ees as it is relatively easy to use at the bedside [31].

4. The left ventricular-arterial coupling (LVAC)

4.1. LVAC and the concept of energy transmission from heart to the proximal arterial tree

The relationship between left ventricular function and the mechanical properties of the proximal arterial tree represents a critical factor to understand cardiovascular performance and energetics. Original studies by Suga [21,22] and Sunagawa [8] laid the groundwork for the concept of LVAC and its assessment through left ventricular P—V relationship analysis. In essence, the P—V diagram of the left ventricle provides insights into the energy transmission from the left ventricle to the proximal arterial tree (Fig. 3, Panel A). Thus, the quadrangular area contained by the P—V loop represents the external or mechanical left-ventricular stroke work, LVSW (i.e., the work produced to eject stroke volume into the pressurized aorta), while the area under ESPVR circumscribed between V_0 and ESV represents the left ventricular potential energy (PE). Meanwhile, the total area circumscribed by the sum of LVSW and PE, i.e. the total pressure-volume area (PVA), represents the total mechanical energy generated by the left ventricle during a cardiac cycle [32–34] (Fig. 3, Panel A), while the LVSW / PVA ratio reflects the bioenergetic efficiency of the left ventricle.

Interestingly, Suga and coworkers demonstrated that PVA is closely related to myocardial oxygen consumption (MVO_2) [35]. Indeed, when MVO_2 is plotted against PVA, a straight line results even under a variety of both pre- and after-load conditions, and contractile states, being the slope of this line an equivalent of the fraction of energy derived from myocardial oxygen consumption that is converted into total mechanical work (Fig. 3, Panel B). Accordingly, the LVSW / PVA relationship should reflect the mechanical efficiency of converting the total energy available (i.e., the PVA) into left-ventricular external work (Fig. 3, Panel A, and B).

4.2. The LVAC assessment/monitoring and its potential clinical value

LVAC reflects the energy transmission from the heart to the proximal arterial tree. Over a range of contractile states, maximal LVSW theoretically occurs when Ea matches Ee (i.e., when Ea = Ees, or, LVAC = 1) [5,6,9]. Meanwhile, maximal myocardial efficiency, that is, the amount of external work performed divided by the myocardial oxygen consumed, occurs when Ea is approximately one-half Ees, during each contractile state (i.e., LVAC ≅ 0.5) [5,6,9]. Remarkably, during shock states, the cardiovascular system might operate at high or low LVAC values (i.e., high or low Ea/Ees ratios), thus suggesting high or low energetic expenditures (Fig. 4). Nevertheless, both high and lower LVAC do not necessarily reflect the adequacy or inadequacy of macrohemodynamics to fulfill tissue metabolic demands, i.e., abnormal LVAC values should not be confounded with the presence or absence of shock.

From a physiological standpoint, any condition in which Ea exceeds Ees (i.e., LVAC >1) could be defined as ventricular-arterial uncoupling [13,36]. Nevertheless, observations in healthy humans have revealed mean Ea/Ees ratios around 1.0 to 1.36 [37], which makes it challenging to identify an exact critical point above which ventricle-arterial "uncoupling" can be established. Despite of such evident limitation, an "uncoupled state" implies that increasing myocardial oxygen consumption will not be translated into improvement of the ventricle work subsequently transmitted to the proximal arterial tree. This might explain why progressively lower LVAC values have been associated with more favorable survival rates in patients with chronic heart failure [38], while progressively higher LVAC values during stress echocardiography might anticipate worse clinical outcomes [39].

However, far beyond static values, variations of LVAC in response to therapeutic interventions could reflect the performance of the cardiovascular system during the hemodynamic resuscitation of shock states. Thus, for example, the infusion of norepinephrine in hypotensive post-operative patients only increased stroke volume in those with an altered LVAC at the baseline, even though arterial hypotension was corrected in

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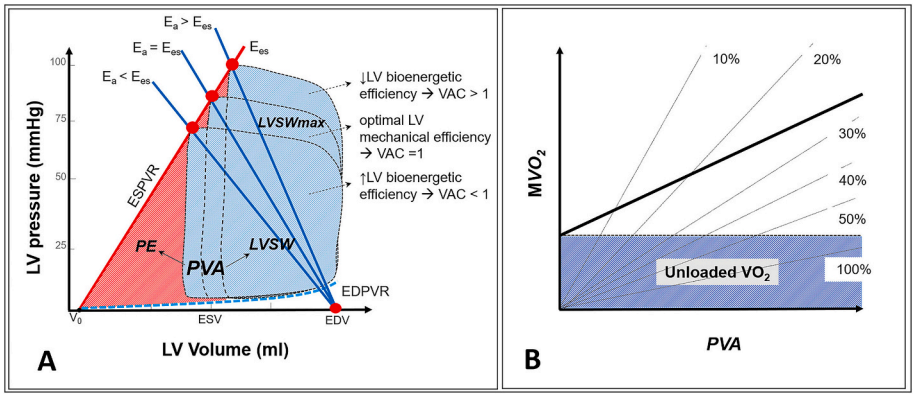

Fig. 3. Theoretical ventricular energetics in the pressure-volume diagram.
Panel A. Pressure-volume area (PVA) is equivalent to the sum of left ventricular stroke work (LVSW) (blue-lined area) and potential energy (PE) (red-lined area). The maximal external or mechanical left ventricular stroke work, LVSW (LVSWmax) should be achieved when Ea = Ees (i.e., LVAC = 1), thus reflecting the maximum mechanical efficiency of the left ventricle. In conditions of Ea < Ees (LVAC <1), left ventricle optimizes its bioenergetic efficiency (i.e., greater LVSW / PVA ratio). Meanwhile, when Ea > Ees (LVAC >1), left ventricle prioritizes the transfer of energy to the periphery (i.e., lower LVSW / PVA ratio); in this last condition, increasing myocardial oxygen consumption does not significantly improve the amount of LVSW.
Panel B. Myocardial oxygen consumption (MVO_2) vs. PVA. The slope of the resultant line represents an equivalent of the fraction of energy derived from myocardial oxygen consumption that is converted into total mechanical work. The MVO_2 under the "X" intercept represents the unloaded MVO_2 (i.e., the myocardial oxygen consumption not related with mechanical contractility processes: basal metabolism, calcium cycling, etc.). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

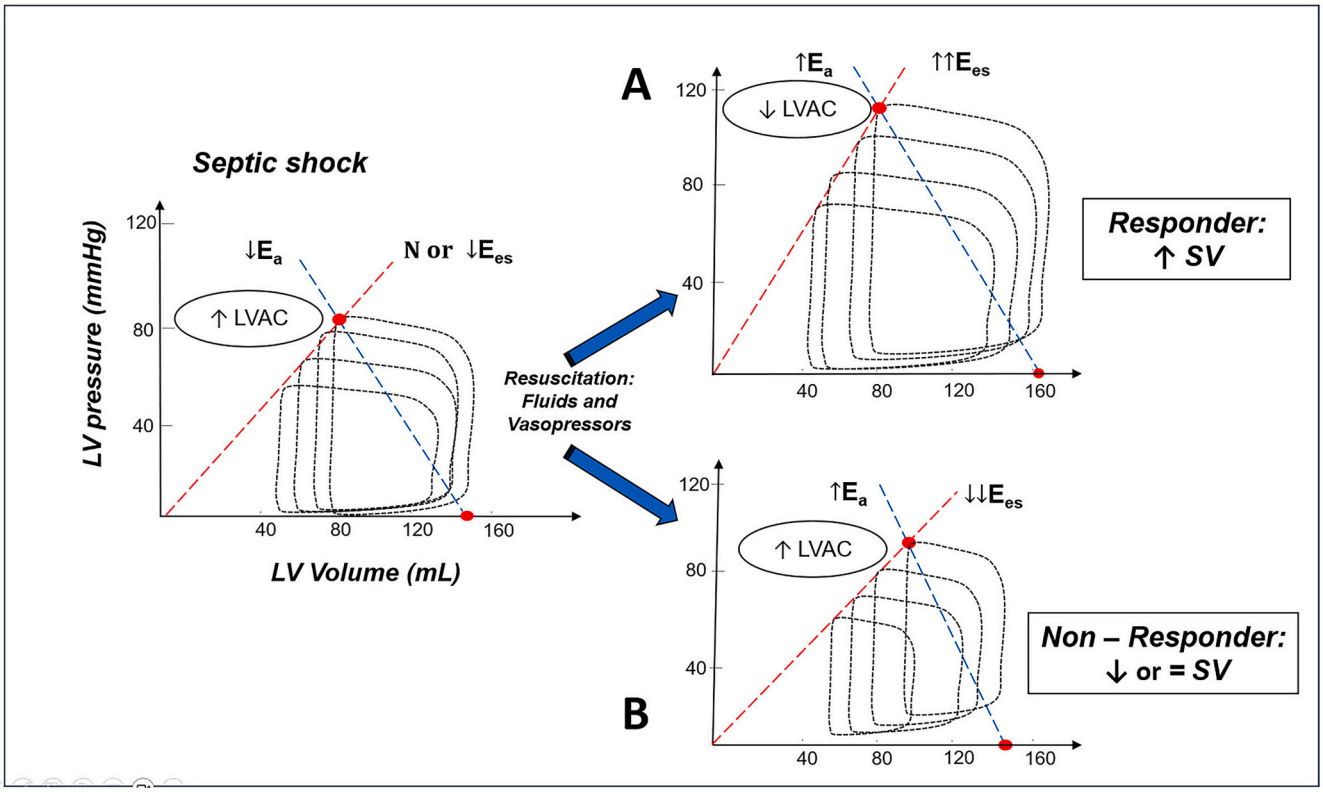

Fig. 4. Hypothetical course of LVAC during initial resuscitation in septic shock.
Two hypothetical scenarios are depicted: **Panel A**. Significant increase in stroke volume (SV), as consequence of predominant increase in Ees and subsequent decrease in LVAC, leading to improve SV; **Panel B.** Non-significant increase in stroke volume, as the increase in Ea it not matched by Ees with a subsequent impairment of LVAC.

the entire cohort [40,41]. Indeed, those increasing stroke volume during norepinephrine infusion showed simultaneous but not symmetric increases in arterial and end-systolic ventricular elastance, with the subsequent decrease in LVAC [40,41]. The impact of norepinephrine on left ventricular afterload should not be neglected. Indeed, while correction of hypotension may restore organ flow, exaggerated increases in ventricular afterload can limit flow generation. Thus, the impact of vasopressor therapy on cardiac output depends on the balance between the direct effects on the heart itself (i.e., increase of coronary perfusion, improvement in cardiac energetics, etc.) and the impact on arterial load, a situation that can be exposed or even anticipated when LVAC is carefully monitored [42,43]. Nevertheless, LVAC assessment is not

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routinely part of conventional hemodynamic monitoring despite recent information highlighting the value of LVAC assessment in resuscitation, prognosis, and management algorithms in diverse clinical contexts [11,13,36–39,44–47]. Conversely, interventions aimed to manage cardiovascular dysfunction in shock states are mainly focused on venous return and cardiac output curves [1,2,43], which neglect the role of arterial load on flow generation and its effect on cardiac performance.

5. Left ventricular-arterial coupling in septic shock

In general, ventricle-arterial uncoupling is common during shock states as reflection of variable degrees of Ea and Ees (Fig. 4). Interestingly, both septic and non-septic vasoplegia may also show increased LVAC values because the coexistence of altered myocardial dysfunction. In addition, therapeutic interventions commonly used in septic shock, such as vasopressors and inodilators may influence on both Ea and Ees, resulting in variable LVAC conditions. Nevertheless, data about dynamic variations and time-course of LVAC in patients with sepsis are limited and come mainly from small-sized cohort studies. Using bedside echocardiography and arterial pressure monitoring, Guarracino and colleagues reported ventricle-arterial uncoupling in 21 of 25 patients with septic shock (mean LVAC of 1.81; IQR: 1.49–2.03) [13]. Subsequently, they studied the effect of different interventions on LVAC in 55 patients with sepsis [44]. Patients with near normal LVAC at baseline showed greater increases in cardiac output (CO) and mean arterial pressure (MAP) after volume loading and norepinephrine initiation compared with those with high LVAC values. Remarkably, LVAC decreased after fluid administration (1.57 ± 0.37 to 1.27 ± 0.36, $p < 0.05$) in those patients in whom CO increased significantly. On the other hand, LVAC increased significantly after norepinephrine administration in those unable to increase SV (1.15 ± 0.38 to 1.80 ± 0.62, $p < 0.05$), or unable to normalize MAP (1.38 SD: 0.44 to 2.12 SD: 0.59 < 0.05) [44]. More recently, Zhou and colleagues analyzed the operative performance of LVAC to predict changes in SV in response to norepinephrine infusion in 38 patients with septic shock. They found that LVAC >1.11 anticipated increases >15% in SV after norepinephrine start (AUC: 0.816 95% CI:0.646–0.927, $p < 0.001$) [45]. As previously described, such response could be explained by the effect of norepinephrine on Ees [42,43]. Interestingly, despite increases in blood pressure, only patients improving LVAC depicted a significant increase in CO after norepinephrine administration [45].

Data about the prognostic value of LVAC suggest the wide heterogeneity of hemodynamic derangements in septic shock. Nasu and colleagues reported that left ventricular-arterial uncoupling frequently occurs in sepsis and its cause —either relative increase of Ea or decreased Ees— varies over the clinical course [46]. Remarkably, a recent study described a U-shaped correlation between LVAC assessed within the first 24 h of ICU admission and clinically relevant outcomes in patients with septic shock, thus suggesting that low LVAC values (as result of combined high Ees and low Ea, during hyperdynamic conditions) are also unfavorable, and consequently, clinical outcomes are not necessarily linked to the cardiovascular bioenergetic efficiency [47].

LVAC assessment could provide valuable information to potentially guide therapy after initial resuscitation efforts (Fig. 5). Thus, for example, inotropic support could be beneficial in patients with persistently low Ees after initial fluid resuscitation and vasopressor support [44,48]. Meanwhile, patients with increased Ea but preserved Ees could theoretically benefit from decreasing vasopressor dose (Fig. 5), although certainly, under close monitoring of tissue perfusion. Nevertheless, information about the clinical impact of LVAC monitoring during resuscitation of septic shock is limited. Recently, Zhou et al. randomized 83 patients with sepsis and evidence of ventricular-arterial uncoupling (defined by LVAC >1.36) to LVAC-guided resuscitation vs. usual care. Patients subjected to LVAC-guided resuscitation had faster lactate

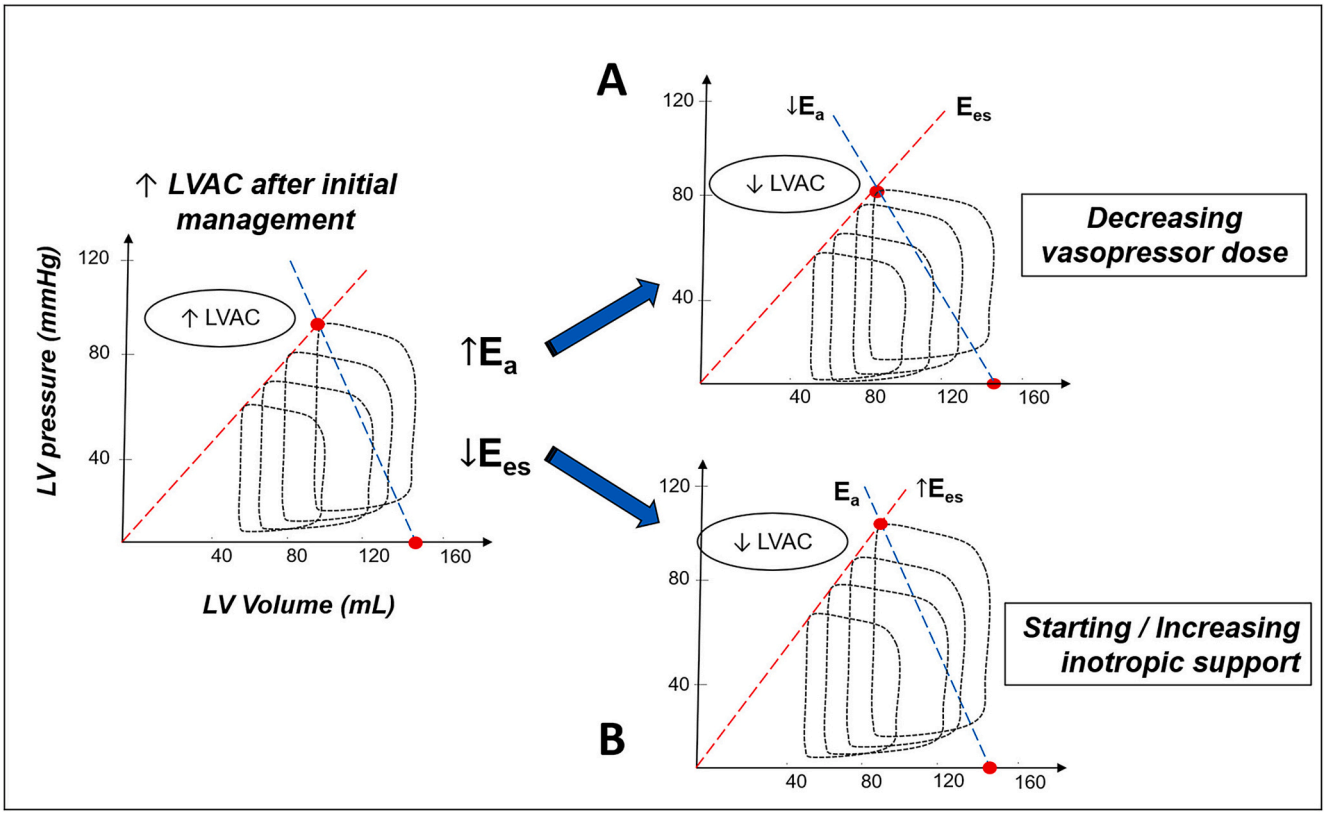

Fig. 5. Hypothetical course of persistently abnormal LVAC after hemodynamic management in septic shock.
Panel A. Increased Ea with preserved Ees. In this case, decreasing vasopressor dose might increase SV; nevertheless, adjustment as this should be performed under close monitoring of tissue perfusion. **Panel B**. Decreased Ees. In this case, starting or adjusting inotrope support should decrease LVAC, thus increasing SV.

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clearance (27.7% IQR: 11.9–45.7 vs 18.3% IQR: 5.7–32.1, $p = 0.038$), although without impact on mortality [48]. Thus, even though physiologically plausible, more information is necessary to estimate the clinical value of LVAC-guided resuscitation in septic shock.

6. LVAC in septic shock: uncertainties and perspectives

Even though there is a growing interest in ventricular-arterial coupling dynamics in critically ill patients, LVAC assessment and its potential utility during shock resuscitation raises conceptual aspects that deserve further exploration. First, the agreement of diverse methods to estimate $Ees_{(SB)}$ is not free of controversy [28,29,49]. In addition, the accuracy and equivalence of non-invasive methods, such as echocardiography or peripheral pulse wave analysis, to estimate ventricular elastance remain questionable [50]. Second, non-invasive estimation of LVAC by echocardiography might be cumbersome as repeated measures are necessary to evaluate LVAC evolvement during the resuscitation process. Third, septic shock might induce central-to-peripheral decoupling in pressure wave propagation [51]. Such phenomenon might lead to an inaccurate estimation of elastances when using peripheral (i.e., radial, femoral) instead of central aortic pressures during conditions of severe vasodilation [52]; although certainly, error would affect almost symmetrically the precision of both Ea and Ees values and consequently, it should not widely affect LVAC calculation itself. Fourth, even though LVAC fundamentals seem to have biological plausibility, there is no convincing evidence that targeting normalization of Ea/Ees ratio will be translated into optimal tissue perfusion, especially under conditions of increased heterogeneity of microvascular flow. Fifth, although a recent small study suggested that LVAC-guided resuscitation could improve lactate clearance [48], hyperlactatemia in sepsis is a complex phenomenon beyond tissue hypoxia [53]. Sixth, even though some data from postoperative cardiac surgery suggest that variations in LVAC correlate with global oxygen consumption [54], interactions between oxygen delivery and consumption in sepsis and septic shock depend on many other complex variables. Thus, while macrohemodynamic resuscitation represents a key element to reverse tissue hypoperfusion in septic shock, LVAC optimization could not guarantee adequate tissue perfusion and oxygenation. Indeed, macrohemodynamics are frequently dissociated from microcirculatory blood flow in septic shock, as it has been widely exposed by other authors [55,56]. Seventh, cardiovascular dysfunction in sepsis and septic shock results from the combination of variable degrees of absolute or relative hypovolemia (because of external fluid losses and increased capillary leakage), pathological vasodilation, myocardial dysfunction, and microvascular blood flow misdistribution. Consequently, LVAC normalization or optimization could be insufficient to reverse the shock process at the cellular level, whereby considerable research efforts should be conducted to elucidate the real clinical value of LVAC assessment to guide therapies during resuscitation of septic shock.

7. Conclusion

Left ventricular-arterial coupling is a physiological concept that integrates arterial and ventricular elastances, allowing for understanding the interactions between heart contractility and the impedance of the proximal arterial tree. This concept provides a holistic view of the relationship between mechanical stroke work and myocardial oxygen consumption, detailing heart contractility and its bioenergetic properties. While LVAC is associated with outcomes in critically ill patients and it has been used to analyze the effect of hemodynamic interventions, there is no current clinical evidence demonstrating that “normalizing” LVAC during resuscitation of septic shock might always reverse tissue hypoperfusion. More detailed studies analyzing the relationships between LVAC and tissue perfusion are required to elucidate the role of LVAC monitoring in patients with sepsis and septic shock.

CRediT authorship contribution statement

Juan Daniel Caicedo Ruiz: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Conceptualization. **Jose Luis Aldana:** Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Conceptualization. **Eduardo Kattan:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Nicolás Orozco:** Writing – review & editing, Writing – original draft, Investigation, Conceptualization. **Gustavo García Gallardo:** Writing – review & editing, Writing – original draft, Investigation, Conceptualization. **Jorge Iván Alvarado Sánchez:** Writing – review & editing, Methodology, Conceptualization. **Juan José Diaztagle Fernández:** Writing – review & editing, Formal analysis, Conceptualization. **Jihad Mallat:** Writing – review & editing, Writing – original draft, Formal analysis, Conceptualization. **Gustavo Adolfo Ospina-Tascón:** Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Formal analysis, Conceptualization.

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Appendix A. Appendix

A.1. Arterial elastance as an approximation of impedance

Arterial elastance (Ea) could be assumed as a surrogate of impedance (Z). According to the three-element Windkessel model, the mean arterial flow ($\overline{Q}$) during one cardiac cycle (T) can be derived from:

$$\overline{Q} = \frac{\overline{P}}{R + Z_c} \tag{1}$$

Since $\overline{Q}$ is equivalent to the ratio between stroke volume (SV) and length of T (i.e., $\overline{Q} = SV/T$), and given that mean arterial pressure equals the area under the pressure curve (A_T) during one cardiac cycle (T), it can be considered that:

$$\frac{SV}{T} = \frac{\frac{A_T}{T}}{R + Z_c} \tag{2}$$

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Then, cancelling for T:

$$SV = \frac{A_T}{R + Z_c} \tag{3}$$

A_T can be substituted by their components (as depicted in the numerator of Eq. 4), in which Pes refers to end-systolic pressure, t_s represents the duration of the systole, t_d represents the diastolic time, and τ (i.e., arterial time constant) equals the product of resistance and compliance, which describes the exponential decay of diastolic pressure [6,16,18]. Hence,

$$SV = \frac{P_{es}\left\{ t_s + \tau\left[1 - exp\left(\frac{-t_d}{\tau}\right)\right]\right\}}{R + Z_c} \tag{4}$$

Which might be rearranged as:

$$\frac{P_{es}}{SV} = \frac{R + Z_c}{\left\{ t_s + \tau\left[1 - exp\left(\frac{-t_d}{\tau}\right)\right]\right\}} \tag{5}$$

Where P_{es} / SV ratio represents the effective arterial elastance, i.e., the Ea.

A.2. Non-invasive estimation of $Ees_{(SB)}$

The algorithm to estimate $Ees_{(SB)}$ assumes a linear end-systolic pressure-volume relation and also supposes a constant volume axis intercept of the end-systolic pressure volume relation (i.e., V_0). First, deriving two values of left ventricular elastance using pressures and volumes determined at times during mid-isovolumetric contraction (t_d) and at end-systole (t_{es}), it is obtained:

$$E_d = P_d/(V_d – V_0) \tag{1}$$

where E_d represents the ventricular elastance at the onset of ventricular ejection point, P_d represents diastolic arterial pressure at the onset of ejection, and V_d represents left ventricular end-diastolic volume.meanwhile,

$$E_{es} = P_{es}/(V_{es} – V_0) \tag{2}$$

Where E_{es} represents the ventricular elastance for the end-systolic point, P_{es} represents end-systolic arterial pressure, and V_{es} represents the left ventricular end-systolic volume.

The E_d / E_{es} ratio (i.e., the E_{Nd}), represents the time and amplitude normalized time varying elastance at t_d. Interestingly, E_{Nd} values are conserved during the isovolumetric period even during cardiac disease, and different heart function and loading conditions (25).

As by definition, $E_d = E_{Nd} \times E_{es}$, then Eq. 1 can be rearranged as:

$$V_d – V_0 = (P_d/E_{Nd})/E_{es.} \tag{3}$$

Combining with Eq. 2, it yields:

$$V_d – V_{es} = [P_d/E_{Nd} – Pe_s)]/E_{es} = [P_d – [E_{Nd} \times {}_{Pes})]/[E_{Nd} \times E_{es}]. \tag{4}$$

which is then solved for E_{es}:

$$E_{es} = [P_d – (E_{Nd} \times P_{es})]/[(V_d – V_{es}) \times E_{Nd}]. \tag{5}$$

Remarkably, left ventricular volume at the onset of ejection (V_d) is closely related to the left ventricular end-diastolic volume, so stroke volume (SV) can substitute for (V_d – V_{es}). In clinical practice, it is accepted that P_{es} approximates systolic arterial pressure (Ps) by the formula:

$$Pes = Ps \text{ x } 0.9. \tag{6}$$

Thus, finally,

$$Ees_{(SB)} = [P_d – (E_{Nd} \times P_s \times 0.9)]/[SV \times E_{Nd}] \tag{7}$$

Appendix B. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jcrc.2026.155486.

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